

# Introduction to Session on Pathology and Epizootiology

by John C. Harshbarger\*

Production of synthetic organic chemicals in the United States began increasing exponentially in the late 1930s and has doubled, on average, every 5 to 6 years from 1.3 million tons to 320 million tons in 1980. Approximately 60,000 compounds are in common usage. As byproducts of this chemical production accumulate in the environment, the impact of the carcinogens and promoters among them are being seen in the fish and shellfish.

In 1964, Clyde Dawe, Mearl Stanton, and Frank Schwartz reported the first liver neoplasms discovered in a population of wild fish—white suckers plus a single case in a brown bullhead—from Deep Creek Lake, Maryland. Dawe and associates speculated on environmental chemicals as the cause. In 1975, Todd Beckerman of the University of Maryland found cholangiomas in white perch collected in Baltimore Harbor and the Chester River (tributaries of the Chesapeake Bay), and the next year, Bruce McCain reported the third population of wild fish—English sole—with liver tumors from Puget Sound, WA. This was followed shortly by John Black's discovery of hepatocarcinomas in sauger and walleye from Torch Lake on the upper peninsula of Michigan and Charlie Smith and associates' discovery of hepatocarcinoma in tomcod from the Hudson River. Today, epizootic liver cancer is known in 15 fish species, all of which are bottom feeders, from approximately 50 polluted sites along both North American coasts, the Great Lakes and tributaries, and other inland waterways. During the same period, experimental tumor induction studies, carcinogen metabolism studies, and DNA adduct studies complemented this historical and epidemiological evidence in suggesting that liver tumors in wild fish result from environmental chemicals.

When this trend became apparent, media interest stimulated Congress to convene a hearing in 1983, which in turn evoked far more media attention. Since the Registry of Tumors in Lower

Animals had been established to build relevant specimen and literature databases by the National Cancer Institute in 1965, at Clyde Dawe's instigation, it was a logical source of information for the media. I estimate that in the past 5 years I have received several thousand requests for information by the media and others regarding fish and shellfish tumors. The number one question is, Is it safe to eat fish with tumors? My answer has always been that the appropriate studies have not been done but that fish tumor cells would probably not grow in a person and that if the fish were cooked, the tumor cells would be unable to grow anyway because they would be dead. I explain further that the residuals of most of the chemicals that are likely to be causing the liver tumors in the fish are stored in the fat and visceral organs which are discarded rather than in the fillet that is eaten. I point out that wild fish are exposed for one to several years before tumors occur, so a person occasionally eating such a fish is ingesting a relatively minute amount of the chemicals that the fish was exposed to unless that person regularly eats fish from the same polluted area over a long period. I suggest that people fish upstream from municipal/industrial complexes, fish in various locations to dilute the risk, that mid-water and surface-feeding species have less chemical exposure than bottom feeding species, and that grilling from underneath further reduces the fat which can store the chemicals. I caution that fish populations with liver tumors are indicating the presence of carcinogens in the environment and there are other routes of potential exposure in addition to eating the fish. These include employment where the chemicals were produced or used, ingesting, breathing, or contact with smoke residues from these sources, and the use of water from the polluted area for drinking, swimming, or bathing.

The five papers in this session deal head-on with the problem of tumors in fish and shellfish related to environmental chemicals and with the question of trophic transfer of carcinogens to consumers.

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